was made. As the temperature fell toward normal the blood pressure dropped from 120/80 to 70/30 mm of mercury. Despite the administration of five liters of fluid, shock persisted, and norepinephrine drip was cautiously started.

The patient was transferred to intensive care where her blood pressure stabilized and she gradually became responsive. She had no seizure activity during her course. Digoxin 0.25 mg and furosemide (Lasix®) 40 mg were given intravenously to support cardiac function and augment urine output. It was soon learned that she had taken 30 tablets of tranylcypromine over the preceding three weeks in addition to five desipramine tablets the past six days. A gas chromatography screening, including salicylates, was negative; two blood cultures were negative and tests of thyroid function were all normal. Creatinine phosphokinase, lactic dehydrogenase, and serum glutamic oxaloacetic transaminase were all elevated but rapidly fell toward normal.

The clinical syndrome could be explained entirely by the interaction between the tranylcypromine and desipramine. The patient improved rapidly except for a few involuntary muscle twitches that also disappeared. Psychiatric consultants believed no brain damage had been done by hyperpyrexia and agreed to observe her as an outpatient upon discharge one week later.

Discussion

The combination of a tricyclic and a monoamine oxidase inhibitor may produce symptoms of dizziness, headache, nausea, vomiting, hyperexcitation, muscle spasm, convulsions, decerebrate rigidity, severe hyperpyrexia, bizarre behavior, and hypo- or hypertension with pronounced individual variation in sensitivity to the combination.[1,3] The severity of the situation must be stressed, since death is not a rare eventuality. Monoamine oxidase inhibitors are thought to produce irreversible inhibition of the enzyme; hence, it is necessary to allow two weeks after cessation of the drug for monoamine oxidase to reaccumulate before administering a drug with a potential interaction.[2] The mechanism of action of Norpramin® (desipramine hydrochloride), a rapidly-acting tricyclic, and of its interaction with a monoamine oxidase inhibitor is unknown. The most common adverse effect seen with the monoamine oxidase inhibitors is the hypertensive crisis in which an indirectly-acting amine (for example, tyramine in certain cheeses) is ingested, this causing acute elevation of blood pressure due to an exaggerated norepinephrine release.[2]

In 1964 tranylcypromine sulfate (Parnate) was temporarily removed from the market because of associated headache, increased blood pressure, and cerebrovascular accidents. An estimated three and a half million persons had taken the drug. In recent years it has been replaced by the tricyclic antidepressants. Monoamine oxidase inhibitors continue in use. For example, phenelzine sulfate (Nardil®) recently was used to suppress rapid eye movement sleep in treating intractable narcolepsy.[4]

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Congenital Absence of Pectoral Muscles

HOMAYOON RAHBARI, M.D.,
Southfield, Michigan

THE ABSENCE OF pectoralis major muscle is probably the most innocuous cause of unilateral hyper-radiolucent lung (Table 1). A recent case of unilateral hyperradiolucent lung proved to be due to this congenital absence.

Report of a Case

The patient was a short, stocky, 30-year-old Caucasian man who was in hospital for acute psychiatric treatment. On a routine chest roentgenogram the radiologist noted left unilateral

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From the Department of Internal Medicine, Kern General Hospital, Bakersfield, Ca.

Reprint requests to: H. Rahbari, M.D., 19381 Dorset, Southfield, Michigan 48075.

TABLE 1.—*Causes of Unilateral Hyper-radiolucence of Lung Field*

A. Extrinsic
   1. Congenital
      a) Absence of pectoral muscles
      b) Sprengel's Deformity
      c) Congenital scoliosis
   2. Acquired
      a) Chest muscles deformity
         1) Atrophy due to disease or disuse
         2) Absence due to surgical excision
      b) Acquired scoliosis

B. Intrinsic
   1. Congenital
      a) Unilateral congenital emphysema
      b) Agenesis or hypoplasia of a pulmonary artery
      c) Agenesis or hypoplasia of lobes of a lung
   2. Acquired
      a) Unilateral emphysema
      b) Thrombosis or embolism of a pulmonary artery
      c) Swyer-James Syndrome*
      d) Unilateral pneumothorax

*A syndrome of uncertain cause thought to be secondary to repeated unilateral lung infections resulting in obliterative bronchitis and bronchiolitis. Collateral ventilation develops distal to the obliteration resulting in secondary overdistention and emphysema. Synonyms: Bronchiolitis obliterans associated with transradiancy, chronic obstructive pseudoemphysema, idiopathic unilateral hyperlucent lung, unilateral hyperlucency of the lung, unilateral nonfunctioning lung. (Swyer PR, James GC: A case of unilateral pulmonary emphysema. Thorax, 1953, 8:133-136)

hyper-radiolucence (Figure 1). The patient had had a left forearm fracture in childhood with no adverse sequelae. He had been working as a construction worker, and gave negative family history regarding the body-build abnormality. The left side of the chest and the left shoulder and upper extremity appeared somewhat smaller than the counterparts (Figure 2). The left side of the chest was almost devoid of hair and perspiration, whereas the right side showed perspiration and considerable hair growth. The left breast was flat and the left nipple was placed more cephalad and laterally than the right. The respiratory and cardiovascular systems appeared normal. After having performed the appropriate maneuvers to decide the extent of the absence of the pectoral muscles (see discussion) it was concluded that this patient had congenital absence of about four-fifths of the pectoralis major and the complete absence of the pectoralis minor on the left side, together with hypoplasia of the left breast and partial absence of the skin appendages on the same side. (See Table 2.)

Discussion

Absence of pectoralis major and minor is not a frequent occurrence, but congenital variations

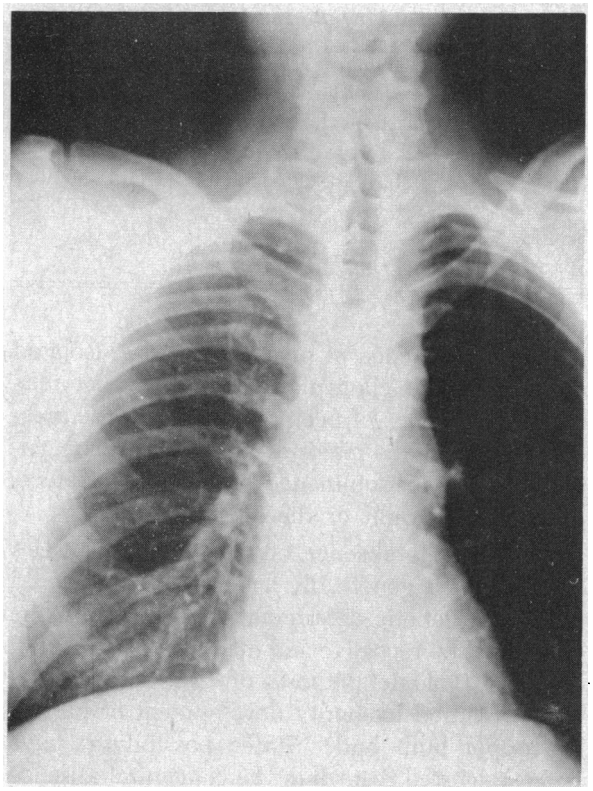

Figure 1.—Hyper-radiolucence on left, found to be due to congenital absence of pectoral muscles.

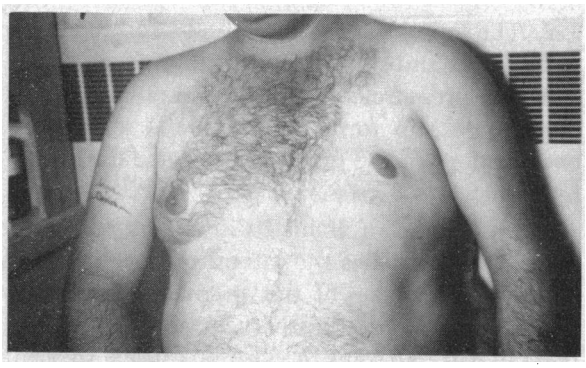

Figure 2.—Differences in hair growth and nipple placement due to absence of pectoral muscles on left side.

occur more frequently in the pectorals than in any other of the skeletal muscles.[1] Often, as in this case, it is the pectoralis minor and the sternocostalis portion of pectoralis major which are missing.[2] The pectoralis major has two origins, the clavicular head and the sternocostal head. Both fleshy heads bend to form a tendon which turns on itself and inserts into the lateral wall of the bicipital groove of the humerus.[3]

TABLE 2.—*Anomalies Associated with Congenital Absence of Pectoral Muscles*

Ipsilateral digital anomalies[2,5,6,7]
Ipsilateral upper extremity shortening[5,6,7]
Absence or hypoplasia of the breast[1,5,6]
Deficiency of chest and axillary hair and sweat glands[1,5]
Defective ribs, costal cartilages, intercostal and shoulder muscles[1,5,6,7]
Scoliosis[6,7]
Sprengel's Deformity[6,7]

Bilateral absence of pectoral muscles, isolated absence of the clavicular portion of pectoralis major, or absence of pectoralis minor in the presence of pectoralis major are rarities.[1] There appears to be no dominance of pectoralis defects on either the right or the left side.[4]

Whether the absence of the pectoral muscles is determined genetically has not been answered clearly; if genetic determination is the case, the gene must be recessive and manifest itself rarely.[5] The embryologic pathosis of this deformity is best explained by faulty development of part of the upper limb bud.[6] Three possibilities have been suggested regarding the congenital absence of pectoral muscles:[1]

1. These structures fail to develop in the embryo.
2. The muscles develop partially, fail to attach to the bone, and subsequently atrophy.
3. The premuscle mass, which in normal development goes to form the pectoralis minor and two portions of the pectoralis major, fails to differentiate into its separate parts.

In an embryo of about 10.5 mm, lateral to the anterior six ribs lies the lateral premuscle mass. The fourth division of this premuscle mass, the pectoral premuscle, passes ventrally to the brachial plexus and joins the arm premuscle sheath.[7] The migration ventro-caudally of this pectoral premuscle mass influences the overlying portions of the skin. In the event of no migration, the mammary gland locates at a higher level and more laterally.[1] It is significant that in the present case the patient could engage in heavy manual labor without any difficulty. It has been noted that those of the living subjects who were in good physical condition showed no inconvenience or awkwardness because of the muscular deficiency.[1] The absence of pectoralis muscles in most cases can be diagnosed clinically. Katz[3] gives the following directions: The subject holds his arms forward a little below the horizontal, and with elbows extended he presses his palms strongly together. This brings the whole muscle into vigorous action, and the two parts, as well as the tendon, can be plainly seen and felt. Then, while the subject is doing this, the observer presses down on the extended arms and instructs the subject to resist the pressure. This instantly causes relaxation of the lower half while the upper half stands out in still stronger action. Conversely, if the observer lifts against the arms and the subject resists, then the upper half relaxes and the lower half acts.

Conclusion

Unilateral hyper-radiolucence of the lung on a roentgenogram merits extensive investigation. One of the simplest reasons for the anomaly is the congenital absence of pectoral muscles.

Summary

The congenital absence of pectoralis minor and sternocostalis portion of pectoralis major on one side is one of the causes of unilateral hyperradiolucent lung. One suggested mechanism for the congenital absence of the pectoral muscles is the failure of the differentiated parts of the pectoral pre-muscle mass in the embryo to become attached to their ultimate insertion sites, and consequently lead to atrophy.

This anomaly can be easily diagnosed clinically by appropriate checks for the function of these muscles.

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